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Relationship between Mean Systemic Filling Pressure, Cardiac Power Output, Myocardial Efficiency and Venous Return Gradient in Liver Transplant Patients

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ABSTRACT

Introduction: The mean systemic filling pressure (PMSF) depends on volemic state, vasomotor tone and cardiovascular function. Therefore, it should be causally correlated with cardiac power, myocardial efficiency and venous return gradient. The aim of the present study was to correlate PMSF with cardiac output, myocardial efficiency and venous return gradient in the perioperative period of patients undergoing orthotopic heart transplantation.

Material and methods. Retrospective, descriptive, analytical cohort study, from January 1, 2021 to January 31, 2022. Inclusion criteria included liver transplantation (ortothopic) adult patients with pulmonary artery catheter. Those with decompensated liver disease due to hemorrhage, grade III ascites or hepatic encephalopathy and those admitted to the ICU without pulmonary artery catheter were excluded. The aim was to correlate PMSF with cardiac power, myocardial efficiency and venous return gradient in the perioperative period of liver transplantation patients.

Results. A strong positive correlation was reported between cardiac power and PMSF just after placement of the pulmonary artery catheter (initial) (r= 0.929, p= <0.001), as well as between cardiac power output and PMSF in the preanhepatic phase (r= 0.591, p= 0.013). For myocardial efficiency strong negative correlations with significant p-value were found for initial PMSF and myocardial efficiency r= -0.659, p= 0.04; between myocardial efficiency and PMSF in the preanhepatic phase r= -0.635, p= 0. 006; myocardial efficiency and PMSF in the anhepatic phase, r= -0.593, p= 0.012; between myocardial efficiency and PMSF in the neohepatic phase r= -0.502, p= 0.040; and for myocardial efficiency and PMSF on admission of patients to the ICU r=-0.571, p= 0.017. Related to the venous return gradient (PMSF - CVP) and PMSF, the correlation was r= 0.919, p= <0.001 (initial) and for the preanhepatic phase with a moderate positive correlation r= 0.490 p= 0.046.

Conclusion. PMSF is useful as a marker for the diagnosis and hemodynamic management of patients undergoing liver transplantation.

KEYWORDS: PMSF, cardiac power output, myocardial efficiency, venous return, liver transplantation.

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I. INTRODUCTION

The mean systemic filling pressure (PMSF) is determined by a large compliant volume of blood coming from the venules and small veins[1]. The normal value of the PMSF varies according to various studies and species, in normal conditions at humans it is between 2-10 mm Hg[2]. It depends on several circumstances: the state of volemia and vasomotor tone. In cardiac surgery and septic shock, values between 15-33 mmHg have been reported, which may be higher due to the administration of liquids or the infusion of vasopressors[3]. The increase in right atrial pressure (RAP) due to the positive pressure received also increases the PMSF which maintains a pressure gradient in relation to venous return[4]. RAP is determined according to the influence of the cardiac pump[5] and maintained by changes in pleural pressure during ventilation which contributes to changes in systolic volume during respiration[6]. The value of RAP that opposes venous return is considered to be the intramaural pressure of the right atrium[7]. The venous system contains approximately 70% of the total blood volume, venous compliance is 40 times greater than arterial compliance, under normal conditions the unstressed blood volume is approximately 70% of the total blood volume, therefore a large reservoir of importance can be recruited by adrenergic venoconstriction. Venoconstriction mobilizes a part of the unstressed volume towards the stressed volume. Venous return according to Poiseuille's law is determined by the resistance to venous return (VRr) and the pressure gradient that exists between the RAP and the venous return is determined by the venous diameter and is controlled by the sympathetic system. The VRr may increase in response to extramural pressure, and two main factors may modify it: the volume of blood mobilized from the venous reservoir (increased by fluid administration) and the capacitance of the venous system which is under the control of adrenergic tone and is determined by the following formula[8]: Venous Return = PMSF - RAP / VRr

Factors that change VRr include increased vascular tone, viscosity and flow redistribution; of these, venoconstriction causes a minimal increase in resistance to venous flow, the main mechanism being the redistribution of blood between different vascular compartments. Venoconstriction decreases the unstressed volume which causes a transient increase in intracardiac pressures such as central venous pressure, mean arterial pressure, conditioning the outflow of this volume to the systolic circulation, being now stressed volume, these changes occur mainly in the splanchnic circulation due to its innervation[9]. Unstressed volume refers to intravascular volume that does not distend the vessel wall, thus not generating intravascular pressure, whereas stressed volume distends the vessel wall causing an increase in intravascular pressure. This generates a positive transmural vascular gradient which is defined by the intravascular and extra thoracic pressures. According to Parkin[10] and Leaning the mathematical estimate is:

PMSF = RAP + MAP + CO

PMSF = RAP x 0.96 + MAP x 0.04 + CO x 0.5

Where RAP = Right Atrial Pressure; MAP = Mean Arterial Pressure and CO = Cardiac Output; 0.96= Constant determined between 0.3 (young) and 1.2 (elderly patient) average 0.5.

Myocardial efficiency (ME) is calculated by the following formula[11]: ME = PMSF - RAP/PMSF, it should be kept in the range of 0 to 1, when the heart falls into asystole the RAP is similar to the PMSF and the myocardial efficacy approaches 0. When myocardial efficacy is less than 0.3, extracardiac mechanical factors should be considered (e.g., pneumothorax, cardiac tamponade).

Hypovolemic shock

During hypovolemia, stressed blood volume decreases along with total blood volume, PMSF decreases while VRr remains unchanged. In the case of preload responsiveness, venous return and cardiac output decrease. In the case of hypotension, this phenomenon is rapidly counter balanced by sympathetic stimulation, which recruits the physiological blood volume reserve without stress, acting as a "volume expansion of its own"[12].

II. MATERIAL AND METHODS

This is a retrospective, descriptive and analytical cohort study. It was conducted in the Intensive Care Unit of the Hospital General de México "Eduardo Liceaga". From January 1, 2021 to January 31, 2022. The sampling was non-probabilistic by convenience. Inclusion criteria included liver transplantation (ortothopic) adult patients with pulmonary artery catheter. Those with decompensated liver disease due to hemorrhage, grade III ascites or hepatic encephalopathy and those admitted to the ICU without pulmonary artery catheter were excluded. The aim was to correlate PMSF with cardiac power, myocardial efficiency and venous return gradient in the perioperative period of liver transplantation patients.

Liver transplantation records for the period from January 1, 2021 to January 1, 2022 were reviewed according to selection criteria. Demographic data were obtained, and pulmonary artery catheter data including the following variables central venous pressure (CVP), mean arterial pressure (MAP), mean pulmonary artery pressure, pulmonary artery occlusion pressure (POAP) and cardiac output (CO) were recorded at 5 times: before surgery just after placement of the pulmonary artery catheter (initial) (1), during surgery (from the transanesthetic record) in three phases: Preanhepatic from incision to portal vein clamping (2). Anhepatic from portal vein clamping to graft reperfusion (3) and Neohepatic (4) from reperfusion to skin closure; as well at patient admission to ICU (5).

To obtain the variable PMSF the formula was used: RAP x 0.96 + MAP x 0.04 + CO x 0.5; Cardiac output was obtained through the Edwards model 774HF75-7.5 F pulmonary artery catheter using the previously described techniques.

Cardiac power output was calculated with the following formula: 0.0022 x MAP x CO; the venous return gradient (VR) was obtained from the following formula: VR = PMSF - RAP.

Myocardial efficiency reported according to the formula EM = PMSF - RAP/PMSF.

Statistical analysis

The information was processed using the SPSS program. The results are presented in frequency distribution graphs to facilitate their evaluation. Descriptive statistics were used to obtain measures of central tendency (median, standard deviation and range for discrete variables, mean and frequencies for nominal variables). The Kolmogorov-Smirnov test was performed.

Comparison of groups for qualitative variables was performed using chi-square or Fisher's exact chi-square. Comparison between quantitative and qualitative variables was performed by t test for related samples or Wilcoxon test according to their distribution. Comparison of means of two or more groups was done by ANOVA (normal distribution) or the Kruskal-Wallis test otherwise; Pearson or Spearman correlation depending on the sample distribution. The value of $p < 0.05$ is taken for statistical significance.

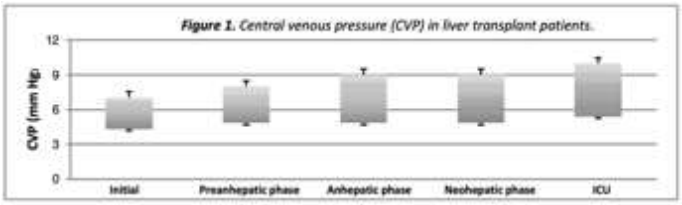
This work was performed with the approval of the institutional research and ethics committees with registration number: DECS/JPO-CT-1242-2022 . The present study is considered without research risk in accordance with the regulations of the general health law on health research.

III. RESULTS

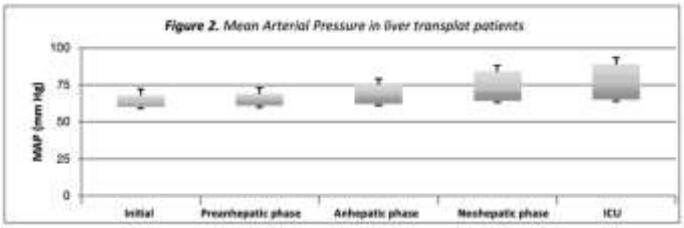
20 files of patients who underwent liver transplantation between January 1, 2021 and January 1, 2022 were reviewed and only 17 met the selection criteria. The rest of the patients (n = 3) were eliminated because the electronic file was not available.

Twelve male (71%) and 5 female (29%) patients were included with a median age of 57 years with an interquartile range (IQR) 15. Of these patients, 12 were male (70.59%) and 29.41%. We also found a median weight of 72 kg (IQR 16), a median height of 1.70 m (IQR 0.18) and a median BMI of 25.46 (IQR 5.06).

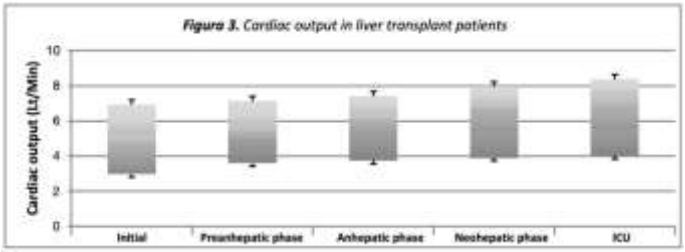
Referring to the measurements obtained through the pulmonary artery catheter, the Central Venous Pressure (CVP) after placement was reported with a median of 5.41 mm Hg (IQR 162); in the preanhepatic phase median of 6.0 mm (IQR 1.59); in the anhepatic phase median of 5. 95 mm Hg (IQR 2.59); in the neohepatic phase median 7.0 mm Hg (IQR 2.05); and at ICU admission median 8.0 mm Hg (IQR 3) *Figure 1*; with significant difference (p= <0. 001) between groups initial phase (time 1), pre-anhepatic (time 2), anhepatic (time 3), neohepatic (time 4) and upon admission to ICU (time 5), when performing the post hoc analysis a significant p-value was reported between the moments CVP 1-CVP 3 (p= 0. 009), CVP 1- CVP 4 (p= <0.001), CVP 1-CVP 5 (p= <0.001), CVP 2-CVP 5 (p= <0.001), CVP3-CVP5 (p= 0.003).



Mean arterial pressure (MAP) at baseline or after catheter placement median 62 mm Hg (IQR 5); in the pre-anhepatic phase median 65 mm Hg (IQR 5); in the anhepatic phase median 68 mm Hg (IQR 5.0); in the neohepatic phase median 71 mm Hg (IQR 4); and at ICU admission median 73 mm Hg (IQR 8) *Figure 2*.

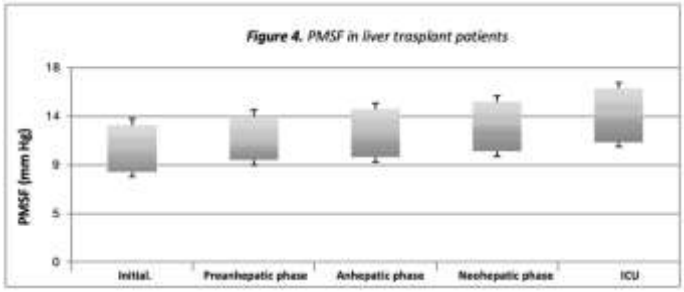


Initial cardiac output was recorded with a median of 4.61 L/min with (RIC 1.50); pre-anhepatic phase of 5.2 L/min (IQR 0.87); median anhepatic phase of 5.89 L/min (IQR 1.14); median neohepatic phase of 6.5 L/min with (IQR 1.62) and at ICU admission median of 6.70 L/min (IQR 1.97) *Figure 3*. Significant difference was found between groups (p= <0.001) and in the post hoc, the groups with p-value < to 0.05 were: initial CO or after placement of the pulmonary artery catheter - CO anhepatic phase(p= 0. 002); initial CO - neohepatic phase CG (p= <0.001); initial CO and CO at ICU admission (p= <0.001); preanhepatic phase CO and CO at neohepatic phase (p= 0.002); preanhepatic phase CO and CO at ICU admission (p= <0.001) and anhepatic phase CO and CO at ICU admission (p= 0.002).

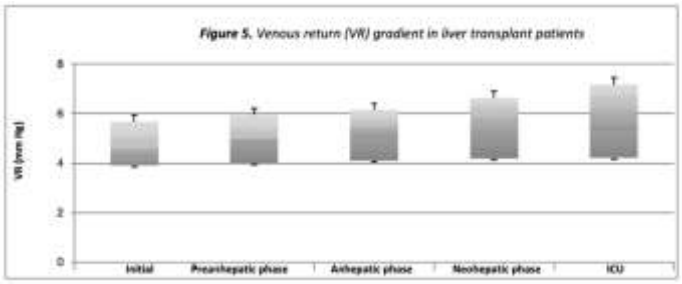


A secondary analysis was performed and reported initial mean systemic filling pressure (PMSF) with median 10.11 mm Hg (IQR 2.04); median preanhepatic phase of 10. 69 mm Hg (IQR1.46); median anhepatic phase of 11.38 mm Hg (IQR 1.81); median neohepatic phase of 12.59 mm Hg (IQR 1.45); and at ICU admission median of 12.95 mm Hg (IQR 1.26) *Figure 4*. With significant difference when comparing the medians of the 5 groups (p=<0.001), in post hoc analysis the groups with significant p-value were initial PMSF-PMSF anhepatic phase (p= 0.001), initial PMSF- PMSF neohepatic phase (p=<0. 001), initial PMSF-PMSF on ICU admission (p=<0.001), PMSF preanhepatic phase- PMSF anhepatic phase (p= 0.003), PMSF

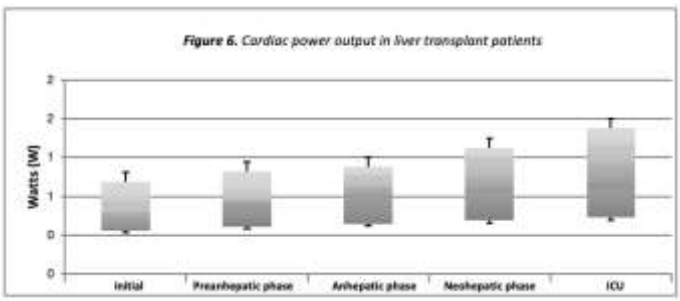
preanhepatic phase- PMSF ICU admission (p=<0.001) and finally PMSF anhepatic phase- PMSF ICU admission (p= 0.003).

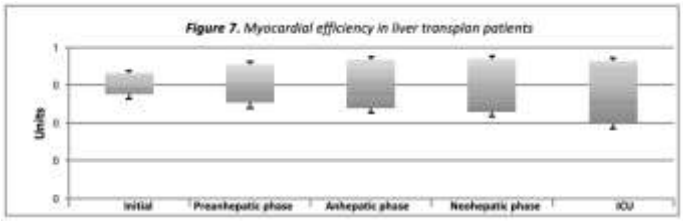


The initial venous return gradient (RV=PMSF-PVC) with a median of 4.59 mm Hg (IQR 0.53); median preanhepatic phase of 5.0 mm Hg (IQR 0.33); median anhepatic phase of 5.43 mm Hg (IQR 0.66); median neohepatic phase of 5.67 mm Hg (IQR 1.01); and at ICU admission median of 6.06 mm Hg (IQR 1.21) *Figure 5*. Comparison between group medians reported a p= <0.001, in post hoc analysis the groups with significant difference included: VR 1-VR 3 (p= 0. 002), VR 1- VR4 (p= <0.001), VR 1- VR 5 (p= <0.001), VR 2- VR 4 (p= 0.002), VR 2- VR 5 (p= <0.001), VR 3- VR 5 (p= 0.002).



The initial cardiac power output (CP) calculated with a median of 0.63 W (IQR 0.17); median preanhepatic phase of 0.75 W (IQR 0.07); median anhepatic phase of 0.89 W (IQR 0.18); median neohepatic phase of 0.97 W (IQR 0.31); and at ICU admission median of 1.10 W (IQR 0.39) *Figure 6*. In this variable, a significant difference was also found when comparing the groups (p= <0.001), when comparing between pairs, the groups with a significant difference were: CP 1-CP 3 (p= 0.002), CP 1- CP 4 (p= <0.001), CP 1- CP 5 (p=<0.001), CP 2-CP 4 (p= 0.002), CP 2- CP 5 (p= <0.001), CP 3- CP 5 (p= 0.002).





Myocardial efficiency (ME) initial or after placement of the pulmonary artery catheter with a median of 0.45 units (IQR 0.05); median preanhepatic phase of 0.46 units (IQR 0.07); median anhepatic phase of 0.47 units (IQR 0.09); median neohepatic phase of 0.44 units (IQR 0.12); and at ICU admission median of 0.42 units (IQR 0.13) *Figure 7*. When comparing the groups in this variable, no difference was found (p= 0.324).

When correlations were performed, a strong correlation was reported between cardiac power and initial PSMF (r= 0.929, p= <0.001), as well as between cardiac power and PSMF in the pre-anhepatic phase (r= 0.591, p= 0.013) *Figure 8*. The rest of the correlations between the cardiac power and PSMF groups in the anhepatic, neohepatic and intensive care unit admission phases were not found to have a p-value <0.05.

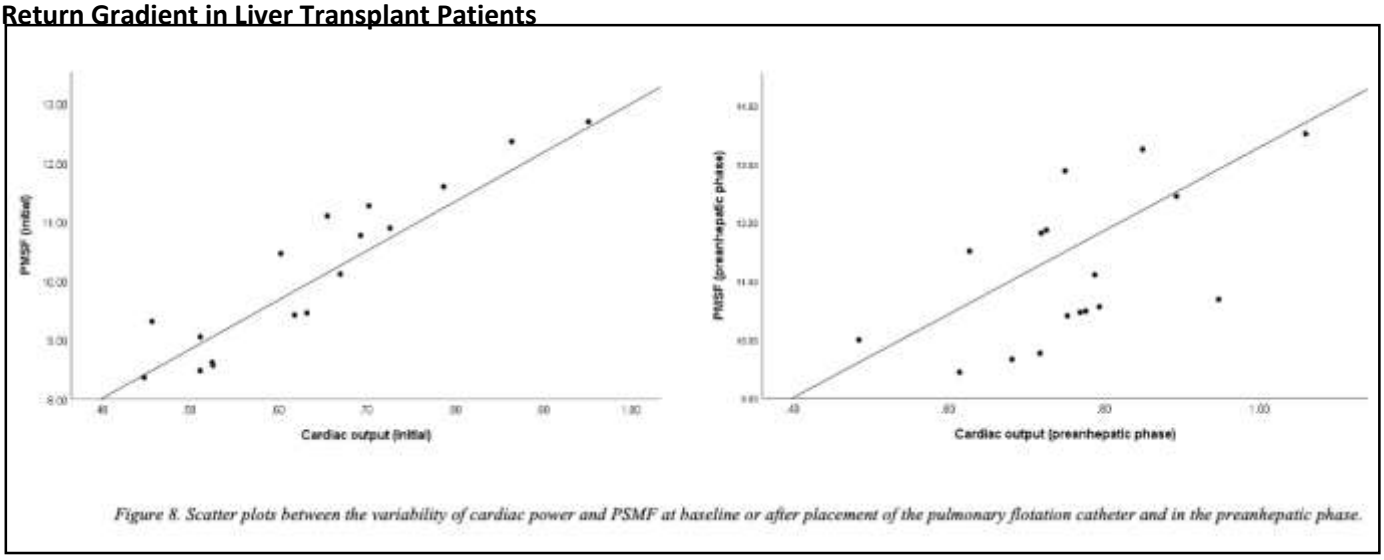


Figure 8. Scatter plots between the variability of cardiac power and PSMF at baseline or after placement of the pulmonary flotation catheter and in the preanhepatic phase.

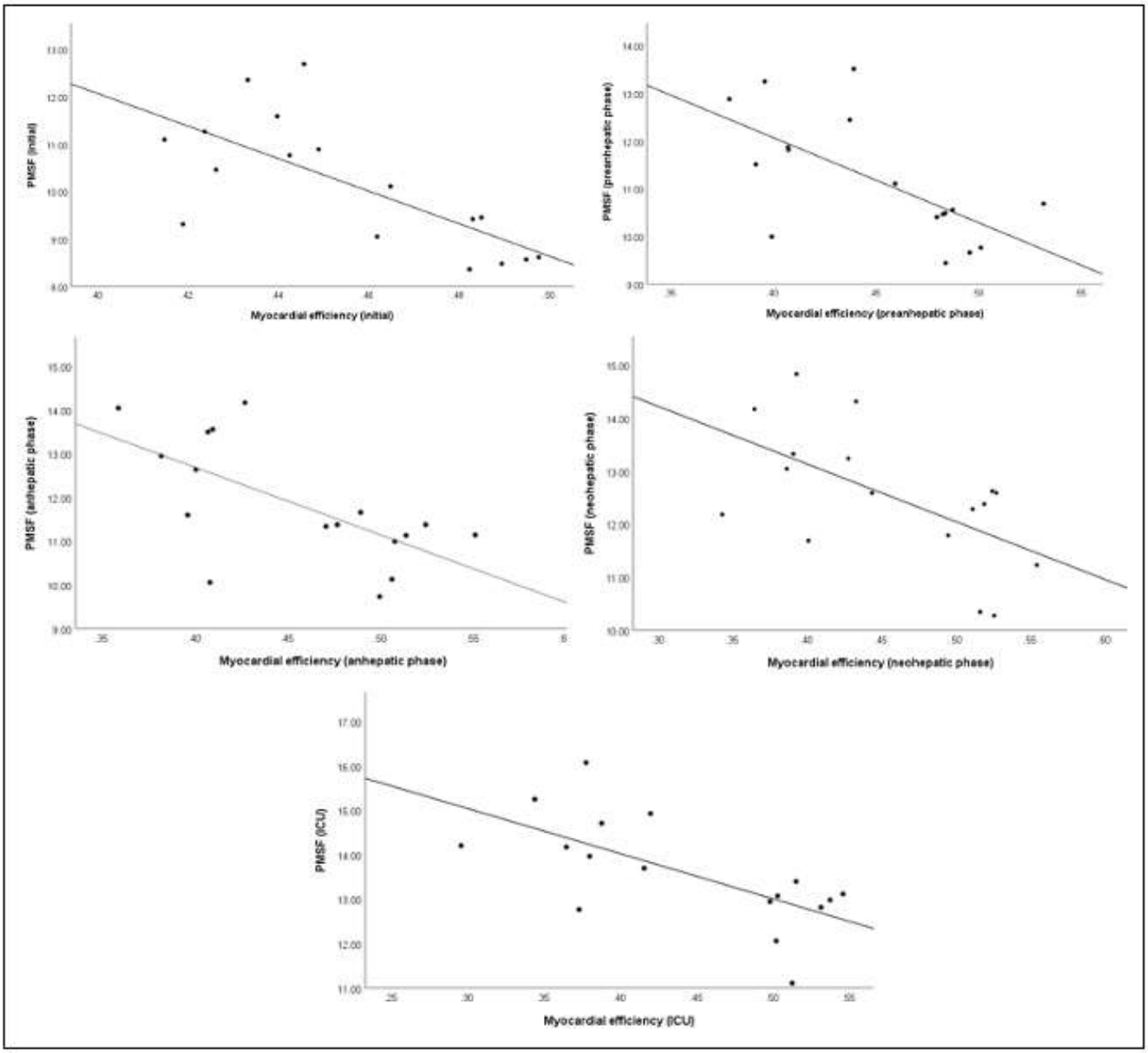


Figure 9. Scatter plots for the variable myocardial efficiency and initial, preanhepatic, anhepatic, neohepatic and on admission of patients to the Intensive Care Unit.

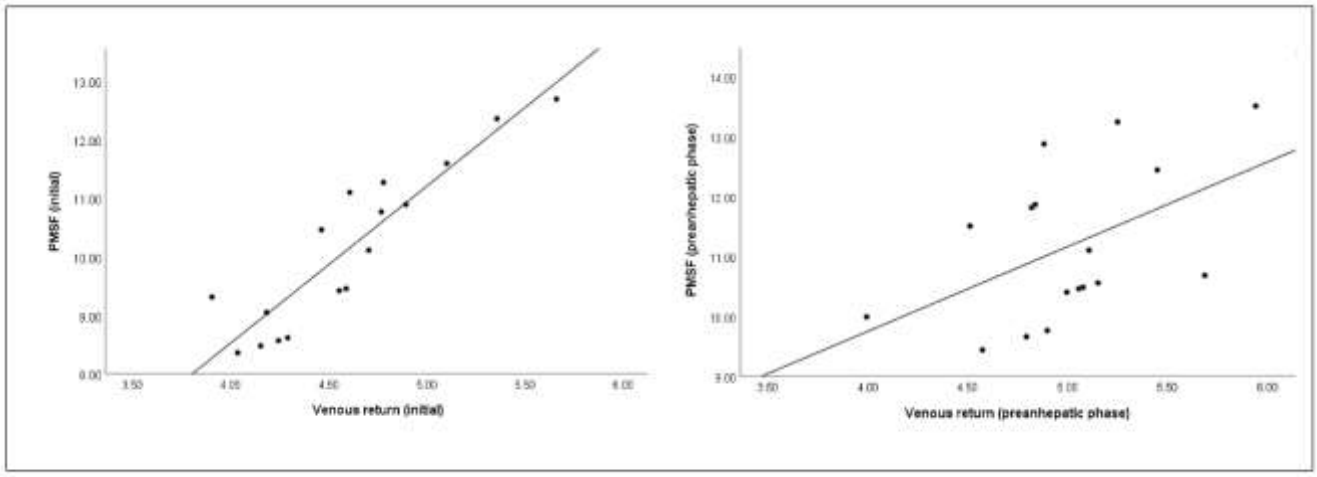


Figure 10. Scatter plots for venous return variable and initial and preanhepatic PSMF.

For myocardial efficiency strong negative correlations with significant p-value were found, as follows: for initial myocardial efficiency and PSMF r= -0.659, p= 0.04; between myocardial efficiency and PSMF in the preanhepatic phase r= -0.635, p= 0. 006; myocardial efficiency and PSMF of the anhepatic phase with an r= -0.593, p= 0.012; between myocardial efficiency and PSMF in the neohepatic phase r= -0.502, p= 0.040; and for myocardial efficiency and PSMF on admission of patients to ICU an r= -0.571, p= 0.017. *Figure 9*

Related to the venous return gradient and PSMF, the correlations found included the initial group with a strong positive correlation r= 0.919, p= <0.001 and in the preanhepatic phase with a moderate positive correlation r= 0.490 p= 0.046; in the rest of the groups (anhepatic phase, neohepatic phase and at ICU admission) no correlations with significant p-value were found. *Figure 10*

IV. DISCUSSION

Seventeen records of liver transplanted patients were analyzed from January 1, 2021 to January 1, 2022.

Liver transplant patients had a median age of 57 years (IQR 48-63 years) being predominantly male and a median weight of 72 kg (IQR 68-84 kg), median height of 1.70 mts (IQR 1.60-1.78 mts) and median BMI 25.46 kg/m2 (IQR 23.51-28.57 kg/m2) considering patients within the range of populations with predominantly good health within their underlying conditions.

Incidence of Diabetes Mellitus was found in 11.76% of patients, systemic arterial hypertension 5.88%, hypothyroidism 5.88%, esophageal variceal disease 88.24% of patients were below the population averages except for variceal disease, which was predominantly found in patients with liver disease requiring liver transplantation.

As for the diagnosis of cirrhosis, it had a prevalence of 94.12% of patients with alcoholic liver disease as a cause of liver transplantation in 41.18%, biliary cirrhosis 17.65%, NADHS 5.88%, NASH 11.76%, Hepatitis C Virus 11.76%, Hepatocarcinoma 11.76%, Portal hypertension 11.76% of patients.

The major determinants of venous return for the regulation of cardiac output are the following factors: blood volume, vasomotor tone, cardiac function (cardiac pump), intrathoracic pressure, body position and right ventricular function.

Central venous pressure trend

At Swan Ganz placement a median of 5.41 mm Hg (IQR 4.87-6.49 mm Hg) was found, with an increase in blood volume at the expense of crystalloid solutions, with adequate intrathoracic pressure, normal cardiac function and decreased vasomotor tone; this leads to an increase in systolic volume which has repercussions on the increase of venous return without increasing the right ventricular end-diastolic pressure (D2VD). During the Preanhepatic phase 6.0 mm Hg (IQR 5.47 -7.0 mm Hg) hypovolemia occurs due to active bleeding with decreased blood volume and compensated by infusion of crystalloids and colloids (albumin), maintaining a stable intrathoracic pressure and a function that still remains normal and a decreased vasomotor tone due to underlying disease.

In the Anhepatic Phase 5.95 mm Hg (IQR 5.41-8.0 mm Hg) a pseudo-normalization of CVP occurs maintaining increased systolic volume with equal venous return increased by transfusion of thermolabile products or autotransfusion of red blood cells, maintaining normal intrathoracic pressure, increasing right ventricular end-diastolic pressure by infusion of vasopressors and decreased vasomotor tone.

In the neohepatic phase 7.0 mm Hg (IQR 5.95-8.0 mm Hg) a congestive phase occurs mediated by an increase in CVP due to increased blood volume, normal intrathoracic pressure, with abnormal cardiac function with persistently decreased vasomotor tone, causing an impediment to adequate diastolic filling of the right ventricle and therefore a progression of the significant increase in CVP found in the values at ICU Admission 8. 0 mm Hg (6.0-9.0 mm Hg) with respect to the start of liver transplantation.

Systolic volume

Systolic volume is directly proportional to cardiac output, which is determined by systolic volume and heart rate (CO=Systolic Volume x Heart Rate). With the use of the Swan Ganz, the following median Systolic Volume (SV) was found: 52.47 ml/beat (IQR 45.27-65.37 ml/beat); with the infusion of crystalloid solutions, cardiac output increases at the expense of preload, with a normal afterload as a result of the difference between end-diastolic volume and end-systolic volume.

In the preanhepatic Phase 67.02 ml/beat (IQR 61.48-74.05 ml/beat) during hypovolemia and volume restitution was normal. In the Anhepatic Phase 75.55 ml/beat (IQR 71.66-82.68 ml/beat) during pseudo-normalization of hemodynamic variables is produced by additional volume restitution and infusion of vasopressors, increases preload-dependent cardiac output, by the increase of volume systolic with an increased afterload due to altered contractile function. In the Neohepatic Phase 87.65 ml/beat (IQR 78.13-92. 29 ml/beat) at graft placement, end-systolic volume decreases due to increased pulmonary vascular resistance, right ventricular end-diastolic volume increases due to increased preload; this conditions an increase in systolic volume and therefore an increase in volume-dependent cardiac output when the afterload is decreased on admission to the ICU 95.94 ml/beat (IQR 85.71-109.71 ml/beat).

Heart Rate

In the case of the Heart Rate (HR): Placement 84 beats/min (IQR 79-89 beats/min) which decreased due to the autonomic dysfunction that patients with cirrhosis present, with chronotropic incompetence and decreased sensitivity of the baroreceptors.

In the preanhepatic phase 80 beats/min (IQR 78-83 beats/min), during the hypovolemia phase and subsequent volume administration, dysfunctional myocardium with impaired contractility to stress and electrophysiological abnormalities is aggravated by increased sympathetic nervous activity.

In the anhepatic phase 78 beats/min (IQR 74-80 beats/min) during the pseudo-normalization of hemodynamic variables the decreased sensitivity to baroreceptors causes a lack of response to vasoconstrictors despite intravascular volume administration so vasopressors are administered during this phase to maintain cardiac function. In the neohepatic phase 75 beats/min (IQR 69-78 beats/min) the neurocardiac abnormalities and autonomic dysfunction cause an increase in cardiac output with subsequent and consequent decrease in heart rate so that on ICU admission 65 beats/min (IQR 58- 75 beats/min) the interquartile range remains similar to the previous one suggesting that most autonomic dysfunctions are potentially reversible after liver transplantation confirming hyperdynamic circulation and portal hypertension as the origin of neurocardiac abnormalities.

Systemic arterial pressure

Mean arterial pressure (MAP) is determined by cardiac output (CO), Central Venous Pressure (CVP) and Systemic Vascular Resistances (SVR). By invasive arterial line monitoring, the median values of the following mean arterial blood pressure (MAP) were found to be significantly increased by crystalloid infusion and subsequent increase in CVP while maintaining lower SVR.

In the preanhepatic Phase 65 mm Hg (IQR 62-67 mm Hg) when hypovolemia occurs it is compensated by infusion of crystalloids and colloids increasing CO and CVP maintaining persistently decreased SVR.

In the anhepatic phase 68 mm Hg (IQR 65-70 mm Hg) the systolic volume, blood and venous return remain increased which increases the CO and CVP with a permanently decreased state in spite of the infusion of vasopressors at low doses.

In the neohepatic phase 71 mm Hg (IQR 69-73 mm Hg) after controlling the bleeding a congestive phase occurs where further increase in CO and CVP with decreased systemic SVR causes adequate impairment of right ventricular diastolic filling which on ICU admission 73 mm Hg (IQR 71-79 mm Hg) is monitored with elevated CO, elevated CVP with normal SVR despite vasopressor infusion.

Cardiac Output/Cardiac Index

The first parameter obtained at pulmonary artery catheter placement was a median of 4.62 L/min (IQR 3.71-5.21 L/min) when infusing crystalloids with the increase of preload increases cardiac output even when ventricular function is depressed. In the preanhepatic phase 5.2 L/min (IQR 4.91-5.78 L/min) there is a compensated hypovolemia with crystalloid/colloid infusion so adrenergic stimulation increases heart rate and produces a positive inotropic effect (dependent on systolic reserve). In the anhepatic phase 5.89 L/min (IQR 5.29-6.44 lt/min) during the pseudo-normalization period, the inotropic effect of catecholamines and/or drugs with positive inotropic effect (norepinephrine) increase cardiac output by utilizing systolic reversal. When the myocardium is found with contractility alterations, the systolic reserve is lower (myocarditis) or lost (necrosis); in these patients the elevation of intraventricular pressure, the systolic stress (afterload) remains normal, which allows a normal ventricular function despite the existence of a pressure overload. In the Neohepatic Phase 6.5 L/min (5.39-7.01 lt/min) when within the ventricle hypertrophy is unable to normalize preload (diastolic stress) as well as afterload (systolic stress) despite normal systolic blood pressure, thus hemodynamic function remains normal despite volumetric overload. On admission to the ICU 6.7 L/min (IQR 5.51-7.48 lt/min) the patient has significant venocapillary hypertension compensated by the activation of the Renin-Angiotensin-Aldosterone axis, which has normalized cardiac output at the expense of the excessive use of the Frank-Starling mechanism mediated by the elevation of intraventricular diastolic pressure (mechanism of impediment to right ventricular filling) vs. mechanism to normalize cardiac output (increased diastolic volume).

Mean Systemic Mean Filling Pressure (PMSF)

In humans the assessment of PMSF by the "Stop-Flow" technique is feasible only in particular situations for real time

measurement, therefore three methods have been determined to be available: The first method takes advantage of the effect of intrathoracic pressure on CO in mechanically ventilated patients during several post-breathing pauses. At different lung volumes the values of CVP and CO are recorded, and the regression line extrapolated to zero flow yields the so-called PMSFhold. Alternatively on the assumption that the same value of intravascular pressure after equilibration will be measured by stopping the flow in the body or in a representative part of it, PMSFhold can be assessed as the intravascular pressure present after 30 seconds of rapid inflation of a cuff placed around the arm at a pressure level above the systolic pressure called PMSFarm. Finally, PMSF has been estimated in terms of mean systemic filling pressure analogously as a parameter extrapolated from three separate measurements, MAP, PVC and GC using coefficients established under the assumption of a fixed relationship between distensibility and arterial and venous resistance. All these assumptions can be transgressed in the patient with cirrhosis and therefore the opinion about the use of this variable is currently biased.

The patient undergoing liver transplantation presents different stages of shock during different hemodynamic phases, as described below21. At Swan Ganz placement, the right ventricle (RV) with normal contractility is subjected to a slight increase in intravascular volume by crystalloid infusion to obtain a greater venous return. In the Pre-Anhepatic Phase, ventricular filling is altered by undergoing an initial hypovolemia with acute compensation with crystalloids and colloids to obtain adequate venous return without affecting D2VD (RV end-diastolic pressure). In the Anhepatic Phase, continuing with the acute increase in right ventricular preload with crystalloids/colloids, transfusion of thermolabile products or autotransfusion produces alterations in contractility and consequently in distensibility, in which case this will be reflected by the increase in RV end-diastolic pressure. In the Neohepatic Phase without hypertrophy, pressure overload may develop in the attempt to normalize RV free wall tension leading to contractile dysfunction as evidenced by the acute clinical overload data present on ICU admission where pressure overload, restriction of the pericardium and the RV and LV shared muscle fiber fascicles limit RV dilatation leading to a greater increase in pressure with less free wall stretch as evidenced by systolic volume, heart rate and cardiac output variables.

The heart can be considered as a hydraulic pump; therefore, cardiac output is defined as the product of the flow and pressure generated by the heart. It is the product of the mean cardiac output and mean arterial pressure determined simultaneously. Cardiac output is not influenced by afterload but can vary directly with preload.

The hemodynamic values measured by Swan Ganz will be modified by the adrenergic response and the resuscitation undergone during the different phases of the trans-surgical procedure. The full understanding of these implications is addressed with the patient on admission to Intensive Care, since it is the critical care physician who integrates, measures and responds to each of the clinical situations presented by the post-transplanted liver patient.

V. CONCLUSIONS

Abrupt increases in pressure or volume over the right ventricle produce changes directly in the right atrium, altering its contraction. During the conduit phase this may be altered due to increased afterload, muscle hypertrophy and late alterations in ventricular relaxation leading to a decreased suction pump phase due to muscle weakness. These decreased reservoir volumes cause a decrease in early diastolic filling and therefore an increase in volume within the right atrium at the onset of atrial systole allowing more effective guidance of fluid response in intensive care by variables such as mean systemic filling pressure.

In the case of the right ventricle (RV), its function is recognized as an important predictor of morbidity and mortality in patients with heart failure. According to guidelines the measurement of right ventricular diastolic and systolic function is reserved for patients awaiting cardiac transplantation, however it has been shown that right atrial (RA) function measured by strain correlates directly with right ventricular diastolic and systolic function, finding a new echocardiographic parameter for the hemodynamic measurement of patients in heart failure.

Limitations of our study included a small sample size, heterogeneity of the etiologies of patients undergoing liver transplantation, differences between patient populations in terms of sex, age and predisposing severity. The studies published to date present statistically significant results and coincide in the line of thought. However, we believe it is necessary to establish a unified standardization of the measurement modality in order to obtain more uniform results and achieve reference values that will help us to identify patients at risk.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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